Colloidal gold, protein, sugar and cells were all found to be within normal limits. Blood creatinine and fasting blood sugar were normal, as were complete hemogram and urinalysis.

Diphenylhydantoin sodium (Dilantin®), 100 mg three times a day was prescribed and in a one month follow-up there were no seizures. The patient then stopped taking Dilantin and at last report no further seizures had occurred. However, it should be noted that the patient had not taken LSD since the occurrence of the seizure, several months before, that had caused him to seek medical consultation.

Discussion

Although this appears to be the first reported case of grand mal seizures associated with ingestion of LSD, it should be noted that many persons with complications of LSD are not seen by physicians or do not go to hospitals.[4] The mechanism by which LSD produced the seizure activity is not known. The drug may have reduced the seizure threshold.

With the continued widespread use of LSD, physicians should be alert to the possibility of yet another side effect from this very controversial drug.

Summary

A man who had no past history of such occurrences, had two grand mal seizures after oral ingestion of LSD. Subsequent neurological and electroencephalographic evaluation showed no abnormalities. Dilantin was prescribed and when at the end of a month no further seizures had occurred, the patient stopped taking the drug. He had not taken LSD since he had the seizure that caused him to seek medical advice.

GENERIC AND TRADE NAME OF DRUG

Diphenylhydantoin sodium—*Dilantin.*

REFERENCES

1. Cohen, Sidney: Personal communication.

2. Cohen, Sidney, and Alpert, Richard: LSD, New American Library, New York, 1966, page 84.

3. Cohen, Sidney: A classification of LSD complications, Psychosomatics, VII:185, May to June 1966.

4. Frosch, W. A., Robbins, E. S., and Stern, M.: Untoward reactions to LSD resulting in hospitalization, New Engl. J. Med., 273:1235-1239, 2 December 1965.

5. Rosenfeld, Albert: The vital facts about the drug (LSD) and its effects, Life, 60:30-31, March 1966.

6. Rosenfeld, Albert: Personal communication.

7. Ungerleider, J. T., and Fisher, Duke D.: LSD: Research and joy ride, The Nation, 16 May 1966.

8. Ungerleider, J. T., Fisher, Duke D., and Fuller, M.: The dangers of LSD, J.A.M.A., 197:389-392, 8 August 1966.

Autoimmune Progesterone Urticaria

THEODORE A. TROMOVITCH, M.D., AND WILLIAM F. HEGGLI, M.D., *San Francisco*

URTICARIA CAN BE caused by a wide variety of factors. The following case report of autoimmune progesterone urticaria stresses that endogenous hormones should be considered in cases of chronic urticaria.

Report of a Case

A 29-year-old white married woman, gravida 4 para 4, had recurrent dermatitis for eight years. The lesions were typical hive-like wheals 0.5 to 3.0 cm in diameter which occurred anywhere on the skin. They regularly appeared seven to 10 days before the menstrual period and spontaneously disappeared approximately the third day past the period. Between times the patient was completely free of dermatitis of any type. Except for this recurrent disease she was in excellent health.

The history did not elicit any relationship of the disease to drugs, and elimination of such common offenders as aspirin and vitamins brought about no remission, nor did elimination diets provide a clue.

A month before she was observed, the temporal pattern of urticaria changed: the lesions continued for a month instead of abating with the cessation of menses. Treatment with antihistamines had given partial relief for a time, but the severity of the eruption had also increased to the point that the use of oral steroids was necessary.

On physical examination the only abnormalities noted were florid bilateral and symmetrical urticarial lesions of the trunk and extremities.

Results of routine examination of blood and urine were within normal limits.

When the lesions were present in a relatively mild state, a single intramuscular injection of 20 mg of progesterone was given. Within an hour the hives already present became larger and new lesions developed over the entire cutaneous surface. The size and number of lesions increased for another hour and it became necessary to stop further progress of the urticaria.

From the Department of Medicine (Dermatology), University of California Medical Center, San Francisco.

Submitted 6 September 1966.

Reprint requests to: 909 Hyde Street, San Francisco 94109 (Dr. Tromovitch).

Premarin® was given on a cyclic schedule, 0.625 mg a day for 21 days each month, to suppress endogenous progesterone production. In addition 15 mg of prednisolone and 32 mg of chlorpheniramine were given daily. Hives continued. At approximately the sixteenth day of the menstrual cycle the dose of Premarin® (conjugated estrogens, equine) was increased to 1.25 mg daily. Within five days urticaria had almost completely disappeared and over the next few weeks the patient was able to discontinue the prednisolone and chlorpheniramine. For three months while taking Premarin 1.25 mg daily she was free of hives. Then a few lesions developed, and at last report the disease was controlled with 1.875 mg of Premarin a day.

Discussion

Shelley and coworkers[2] recently described autoimmune progesterone dermatitis. Their patient was a 27-year-old woman who, at the time of menses, had an extensive vesicular and bullous eruption resembling dermatitis herpetiformis clinically and erythema multiforme histologically. Flares of the dermatitis could be regularly produced by injection of progesterone and progestational compounds. Several hours after a single dose of progesterone, the characteristic lesions would develop and show degranulation with disappearance of circulating basophil leukocytes. Clinical remission could be induced by cyclic oral administration of ethinyl estradiol, 0.05 mg daily. Following oophorectomy the patient had complete involution of all clinical signs and symptoms.

Zondek and Bomberg[3] in reviewing endocrine allergy also described a case of progesterone urticaria. Both they and Kupperman[1] discussed skin testing with hormones. However, skin testing gives variable results. Direct production of urticaria by intramuscular injection of progesterone proved to be a definitive test in the case herein reported and in one reported by Shelley and coworkers.[2] The patients in both cases may well present part of a spectrum. In the present case, the appearance of urticaria within one hour after injection of 20 mg of progesterone suggests an immediate hypersensitivity reaction. Shelley's case, in which there was reaction of erythema multiforme type and a latent period of approximately seven hours after injection of the progesterone, suggests an intermediate hypersensitivity.

Summary

In a case of autoimmune progesterone urticaria, the patient had typical urticaria for the week before menstruation and through the menstrual period, with complete freedom of eruption in the intervening time. This persisted for eight years. In tests, the lesions were found to occur within an hour after administration of 20 mg of exogenous progesterone. Oral administration of Premarin, 1.25 to 1.875 mg a day, in cyclic fashion, controlled the condition.

GENERIC AND TRADE NAME OF DRUG

Conjugated estrogens (equine)—*Premarin.*

REFERENCES

1. Kupperman, H. S.: Human endocrinology, F. A. Davis Co., Philadelphia, 1963.

2. Shelley, W. B., Preucel, R. W., and Spoont, S. S.: Autoimmune progesterone dermatitis, J.A.M.A., 190:35-38, 5 October 1964.

3. Zondek, B., and Bomberg, Y. M.: Endocrine allergy: Clinical reaction of allergy to endogenous hormones and their treatment, J. Obs. & Gyn. Brit. Comm., 54:1-19, 1947.

Amebic Granuloma Of the Cecum

WALTER M. SHAW, M.D., *Chula Vista,* AND BENEDICT R. WALSKE, M.D., *Tucson, Arizona*

INFREQUENTLY, INTESTINAL amebiasis may be manifested by a granuloma which is an end result of destruction and repair with an overgrowth of granulation tissue. Microscopically, the granuloma shows fibroblasts, collagen, chronic inflammatory debris and necrosis and is located chiefly in the submucosal and the subserosal layer.[7] The rectum and cecum are the most common sites for these granulomas.[5]

This lesion may mimic carcinoma and it is difficult and sometimes not possible to differentiate between an amebic granuloma and a carcinoma of the colon. The deformity of the colon presented by most neoplasms is more likely to be asymmetrical as compared with the more characteristic symmetrical distribution of defects in the intestinal wall caused by amebiasis.[2] It may, of course, not be possible to distinguish the difference. The differential diagnosis will also include consideration of

From the Surgical Service, Veterans Administration Hospital, Tucson, Arizona.

Submitted 10 October 1966.

Reprint requests to: McCausland-Robinson Clinic, 275 F Street, Chula Vista 92010 (Dr. Shaw).